UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF NEW JERSEY
CAMDEN VICINAGE

IN RE: VALSARTAN, LOSARTAN, AND IRBESARTAN PRODUCTS LIABILITY LITIGATION **THIS DOCUMENT RELATES TO:** *Gaston Roberts et al. v. Zhejiang Huahai Pharmaceutical Co., et al.*, Case No. 1:20-cv-00946-RBK-JS	MDL No. 2875 Honorable Renée Marie Bumb District Court Judge

DEFENDANTS' MEMORANDUM IN OPPOSITION TO PLAINTIFF'S MOTION TO EXCLUDE THE OPINIONS OF NADIM MAHMUD, M.D., M.S., M.P.H., M.S.C.E.

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INTRODUCTION

Dr. Nadim Mahmud is a prominent hepatologist at the University of Pennsylvania. In a 40-page report containing more than 100 citations to scientific literature and other medical sources, Dr. Mahmud carefully "respond[s] to the opinions offered by" Plaintiff's expert, Dr. Fareeha Siddiqui, "regarding the potential causes of" Gaston Roberts' hepatocellular carcinoma ("HCC") and provides his "independent expert opinion regarding the cause or causes of Mr. Roberts' cancer." (Rep. of Dr. Nadim Mahmud at 1 (Pl.'s Br. Ex. B).) Although Plaintiff urges the Court to "exclude Dr. Mahmud from testifying at trial" (Pl.'s Br. at 39), Plaintiff ***does not*** address the vast majority of Dr. Mahmud's opinions, including the following:

- The scientific literature establishes that cirrhosis of the liver, which afflicted Mr. Roberts, is the most "well-known and well-established risk factor for HCC" (Mahmud Rep. at 2-3, 18-19);

- Dr. Siddiqui's opinion that Mr. Roberts' cirrhosis was supposedly "mild" is contrary to science (*id.* at 35-36);

- NDMA exposure is not recognized as a risk factor for HCC within the hepatology community because the only two epidemiologic studies of valsartan users reported, at most, a very weak association, and an evaluation of the Bradford Hill factors weighs strongly against an inference of causality (*id.* at 3, 25-32);

- Dr. Siddiqui failed to reliably rule out several potential causes of Mr. Roberts' HCC, including his obesity, metabolic dysfunction-associated steatotic liver disease ("MASLD"), and diabetes (*id.* at 3, 23-24); and

- Dr. Siddiqui's reliance on animal studies, studies addressing diseases other

> than liver cancer, and studies involving occupational exposures to support her conclusion that Mr. Roberts' valsartan use was the only substantial factor in causing his HCC is highly unscientific (*id.* at 30-37).

By failing to challenge the core of Dr. Mahmud's opinions, Plaintiff has effectively conceded that his bottom-line testimony is admissible. While Plaintiff does criticize other opinions proffered by Dr. Mahmud, her arguments are meritless.

First, Plaintiff argues that Dr. Mahmud's opinion that Mr. Roberts had cirrhosis well before his August 2018 HCC diagnosis is inadmissible because it is based "solely" on a "Fib-4" score that Dr. Mahmud calculated for Mr. Roberts. That is not true. Dr. Mahmud offered several bases for his opinion that Mr. Roberts had cirrhosis by April 2016, including unequivocal statements by both parties' radiology experts supporting his opinion. In addition, Mr. Roberts' FIB-4 scores—calculations that also show pre-existing cirrhosis was likely—further support Dr. Mahmud's opinion that Mr. Roberts had preexisting cirrhosis. Plaintiff's criticisms of Dr. Mahmud's reliance on those scores misunderstand the burden of proof, distort his testimony and, at most, raise questions for cross-examination.

Second, Plaintiff seeks to preclude Dr. Mahmud from criticizing Dr. Siddiqui's extreme, outlier opinion that NDMA exposure is a "cause of cirrhosis." (Pl.'s Br. at 34-35.) Plaintiff insists that Dr. Mahmud should have credited Dr. Siddiqui's dubious theory based on a 70-year-old anecdotal case report involving two individuals, but case reports cannot demonstrate an association, much less a

causal relationship, between NDMA exposure and cirrhosis.

Third, Plaintiff argues that Dr. Mahmud improperly criticized Dr. Siddiqui's rejection of Mr. Roberts' longstanding history of metabolic dysfunction-associated steatohepatitis ("MASH") (previously known as "NASH" in the medical community) as a factor in causing his HCC because there is no evidence of a formal diagnosis of this condition. But, as Dr. Mahmud explains, Mr. Roberts' history of MASH is well-supported by his medical records, including his treating physicians' view that he had "HCC arising out of NASH cirrhosis[.]" (GRobertsJr-UABHIM-MD-00085 (Ex. 1 to Decl. of Nina Rose ("Rose Decl.")).)

Fourth, Plaintiff argues that Dr. Mahmud gave "additional specific causation opinions" not disclosed in his report when he testified that NDMA may have a protective effect on liver health. (Pl.'s Br. at 35.) But Plaintiff misses the point of Dr. Mahmud's testimony, which was merely to highlight that the animal studies relied upon by Dr. Siddiqui all involved normal livers as baselines. As Dr. Mahmud explained, this is one more reason why Dr. Siddiqui's attempt to extrapolate the animal data to humans with cirrhosis is flawed.

Fifth, Plaintiff argues that Dr. Mahmud improperly relied on an unreliable Tumor Volume Doubling Time calculation and Defendants' radiology expert Dr. Victoria Chernyak in opining that Mr. Roberts' HCC may have predated his first exposure to valsartan potentially contaminated with NDMA. But Dr. Mahmud's

doubling-time opinion is grounded in the scientific literature and in basic statistical principles. Moreover, it was entirely appropriate for Dr. Mahmud (who routinely interprets radiological imaging) to note that his own views are supported by the expert review of Dr. Chernyak, a highly qualified radiologist with significant expertise in identifying HCC based on imaging.

Finally, Plaintiff asserts at the very end of her brief that Dr. Mahmud should be broadly barred from opining "as to the [c]ause of Mr. Roberts' [l]iver [c]ancer" because his views are purportedly based entirely on the belief that "Mr. Roberts had liver cancer prior to ingesting" valsartan potentially contaminated with NDMA. (Pl.'s Br. at 37-39.) This is not true. Dr. Mahmud's opinion is rooted in the fact that Mr. Roberts has numerous, well-established risk factors for HCC, including his cirrhosis, MASLD/MASH, obesity, and diabetes. (Mahmud Rep. at 3, 17-41.) The existence of "strong evidence that Mr. Roberts' HCC may have been present" before he ever took a valsartan pill potentially contaminated with NDMA only reinforces the soundness of his opinions. (*Id.* at 3.)

BACKGROUND

Dr. Mahmud graduated from Stanford University School of Medicine in 2013, after which he completed a residency in internal medicine, as well as a gastroenterology fellowship and an additional fellowship in transplant hepatology. (Mahmud Rep. at 1.) "Since June of 2020, [Dr. Mahmud] ha[s] worked as a faculty

Transplant Hepatologist at both the Hospital of the University of Pennsylvania and at the Corporal Michael J. Crescenz Philadelphia Veterans Affairs Medical Center[.]" (*Id.* at 2.) In this capacity, Dr. Mahmud "care[s] for patients across the spectrum of chronic liver disease" and has "participated in the care of hundreds of patients with HCC[.]" (*Id.*) This experience has afforded Dr. Mahmud "significant knowledge and expertise with respect to the risk factors that contribute to the development of liver cancers such as HCC." (*Id.*)

Dr. Mahmud has published more than 145 peer-reviewed publications, "including studies addressing HCC diagnosis, management, and outcomes[.]" (*Id.*) He has also "given invited clinical and research talks at major international liver conferences, including the American Association for the Study of Liver Diseases (AASLD), the European Association for the Study of the Liver (EASL), and the Asian Pacific Association for the Study of the Liver (APASL)." (*Id.*)

Dr. Mahmud "respond[s] to the opinions offered by" Dr. Siddiqui, and in particular, her "differential diagnosis." (*Id.* at 1, 3.) As he explains, Dr. Siddiqui lacked a reliable basis to "rule in" NDMA as a cause of Mr. Roberts' HCC because the epidemiology is weak, inconsistent and contains a number of limitations and biases. (*Id.* at 3, 27-32.) Dr. Mahmud also details all of the reasons why animal studies exposing rodents to large amounts of NDMA cannot be reliably extrapolated to humans. (*Id.* at 30-33.) And Dr. Mahmud addresses the key Bradford Hill factors

of strength, consistency, dose-response, and temporality, which he concludes are not satisfied and do not support an inference of causality. (*Id.* at 3, 33.)

Dr. Mahmud also explains that Dr. Siddiqui lacks any scientific basis to "rule out" Mr. Roberts' numerous known causes of HCC, including, most notably, cirrhosis of the liver, which "is the most well-known and well-established risk factor for HCC." (*Id.* at 2.) In so explaining, Dr. Mahmud goes through the scientific literature, which shows that patients with cirrhosis, and in particular cirrhosis related to fatty liver disease, "have a 30- to 45-fold increased risk of developing HCC, relative to patients without cirrhosis." (*Id.* at 3.) Dr. Mahmud also explains that Dr. Siddiqui improperly brushes aside Mr. Roberts' obesity, MASLD/MASH and diabetes—all of which are independently associated with significant risks of HCC. (*Id.* at 3, 18-24, 35-40.) Finally, Dr. Mahmud explains that there is "strong evidence that Mr. Roberts' HCC may have been present" before he ever took valsartan potentially contaminated with NDMA and criticizes Dr. Siddiqui for failing to consider that strong possibility. (*Id.* at 3, 33-34.)

ARGUMENT

Rebuttal experts addressing causation have no "burden" to affirmatively disprove causation. *See Holbrook v. Lykes Bros. S.S. Co.*, 80 F.3d 777, 786 (3d Cir. 1996) (rebuttal expert may opine on possible alternative causes without certainty that they caused the plaintiff's injury because disproving causation is "a burden

which the defense d[oes] not bear"). Instead, it is "entirely appropriate" for defense experts to offer what are "essentially, critiques of [p]laintiffs' experts' evidence, methodologies, and conclusions." *In re Abilify (Aripiprazole) Prods. Liab. Litig.*, 299 F. Supp. 3d 1291, 1368 (N.D. Fla. 2018); *see also Feliciano v. CoreLogic Saferent, LLC*, No. 17-cv-5507 (AKH), 2020 U.S. Dist. LEXIS 199069, at *8 (S.D.N.Y. June 11, 2020) (defense expert can simply "pok[e] holes in [other side's] argument").

Plaintiff's motion ignores this standard. To the extent she cites a handful of cases in a section labeled "Rule 702 and *Daubert* Standards" (Pl.'s Br. at 6-8), nearly all of them excluded ***plaintiffs'*** experts who failed to follow their own stated methodology in forming affirmative opinions on the issue of general or specific causation. *See, e.g.*, *In re Paoli R.R. Yard PCB Litig.*, 35 F.3d 717, 770-71 (3d Cir. 1994) (cited in Pl.'s Br. at 7); *In re Zoloft (Sertraline Hydrochloride) Prods. Liab. Litig.*, 858 F.3d 787, 800 (3d Cir. 2017) (cited in Pl.'s Br. at 6); *Elcock v. Kmart Corp.*, 233 F.3d 737, 750 (3d Cir. 2000) (cited in Pl.'s Br. at 7-8). And in the one case she cites involving a defense expert, the court ***denied*** the motion because the expert's testimony was "based upon his own extensive knowledge of the peer-reviewed medical literature. . . . his review of the data . . . [and] his decades of clinical experience . . . ." *Magistrini v. One Hour Martinizing Dry Cleaning*, 180 F. Supp. 2d 584, 612 (D.N.J. 2002) (cited in Pl.'s Br. at 7), *aff'd,* 68 F. App'x 356 (3d Cir.

2023).

Here, applying his years of clinical practice and citing to numerous scientific papers and Mr. Roberts' medical records, Dr. Mahmud meticulously criticizes Dr. Siddiqui's departures from established science, both in "rul[ing] in" NDMA in valsartan as a potential cause of Mr. Roberts' HCC and in "rul[ing] out" Mr. Roberts' numerous well-established and significant risk factors, including his cirrhosis, MASLD/MASH, obesity, and diabetes. (*See* Mahmud Rep. at 14-15; *see also id.* at 14-41.) Plaintiff does not address the core of these opinions. And while Plaintiff challenges various snippets in Dr. Mahmud's report, none of her arguments justifies exclusion of any of his opinions.

I. DR. MAHMUD PROPERLY CRITICIZES DR. SIDDIQUI'S OPINION THAT MR. ROBERTS' HCC WAS NOT CAUSED BY CIRRHOSIS.

As Dr. Mahmud explains, Dr. Siddiqui's attempt to "rule out" Mr. Roberts' cirrhosis as a likely cause of Mr. Roberts' HCC by labeling it "mild" is unreliable because such a concept is not recognized in the hepatology community. (Mahmud Rep. at 3, 35-38.) Plaintiff does not address this core opinion. Instead, she complains that "Dr. Mahmud plans to tell the jury that Mr. Roberts had cirrhosis years before his cancer diagnosis, based solely on predictive modeling and subjective interpretation." (Pl.'s Br. at 34.) This argument mischaracterizes Dr. Mahmud's opinions. As Dr. Mahmud points out, even Dr. Siddiqi acknowledges that Mr. Roberts had cirrhosis in 2016. (Mahmud Rep. at 35 (citing Expert Report of Fareeha

Siddiqui, M.D. ("Siddiqui Rep.") at 30, Mar. 10, 2025) (Rose Decl. Ex. 2)).) Moreover, ***both*** Plaintiff's and Defendants' radiology experts agree that an April 2016 CT scan reflects "'an enlarged liver . . . observed in patients with underlying cirrhosis.'" (*Id.* at 22 (citing Rep. of Dr. Christopher Mele ("Mele Rep.") at 2); *see also id.* (citing Rep. of Dr. Victoria Chernyak at 3-4).) Plaintiff does not seriously dispute any of this evidence, which clearly constitutes "'good grounds' for [Dr. Mahmud's] proposed testimony" regarding the timing of Mr. Roberts' cirrhosis. *See Winn-Dixie Stores, Inc. v. E. Mushroom Mktg. Coop.*, No. CV 15-6480, 2021 WL 2352016, at *14 (E.D. Pa. June 9, 2021). Rather, Plaintiff appears to be suggesting that Dr. Mahmud should have ignored this evidence because "Mr. Roberts never underwent a liver biopsy necessary to diagnose [him] with cirrhosis." (Pl.'s Br. at 34.) However, as Dr. Mahmud explained at his deposition, "these days [hepatologists] don't need to rely on biopsies because we have very well-validated ways of identifying cirrhosis just based on labs, imaging, prediction models, et cetera." (Dep. of Nadim Mahmud ("Mahmud Dep.") 110:14-21 (Pl.'s Br. Ex. A).) This is not just Dr. Mahmud's opinion; it is the view held throughout the hepatologic community.[1]

[1] *See, e.g.*, Richard K. Sterling et al., *AASLD Practice Guideline on noninvasive liver disease assessment of portal hypertension*, 81 Hepatology 1060, 1063 (2024) (Rose Decl. Ex. 3) (discussing use of "[f]eatures of general imaging studies to diagnose cirrhosis and portal hypertension"); Perry J. Pickhardt et al., *Multiparametic CT for Noninvasive Staging of Hepatitis C Virus-Related Liver*

Plaintiff also challenges Dr. Mahmud's statement that additional factors further suggest Mr. Roberts' cirrhosis likely existed years before his HCC diagnosis, specifically: (1) the 2016 CT scan showing an enlarged spleen, a classic cirrhosis symptom; and (2) Mr. Roberts' "FIB-4" scores, which are also consistent with pre-existing cirrhosis. (Pl.'s Br. at 10, 21-33.) Neither challenge has merit.

A. Dr. Mahmud's Opinion That Mr. Roberts Had An Enlarged Spleen In 2016 Is Admissible.

Plaintiff argues that Dr. Mahmud "lack[s] the radiological expertise" to conclude that Mr. Roberts' "'spleen size is enlarged'" (a classic cirrhosis symptom) based on a 2016 CT. (Pl.'s Br. at 11 (citing Mahmud Rep. at 22).) Plaintiff's focus on Mr. Roberts' spleen size ignores Dr. Mahmud's discussion of multiple other aspects of the 2016 CT that "clearly demonstrate[] the presence of cirrhosis"—i.e., the "overtly nodular" appearance of the left hepatic lobe, the enlargement of that lobe and the caudate lobe, and the recanalization of the umbilical vein. (Mahmud Rep. at 21.) But even as to Plaintiff's narrower challenge, Dr. Mahmud explains that Plaintiff's own expert radiologist, Dr. Mele, specifically described the April 2016 scan as depicting an "'enlarged spleen[.]'" (Mahmud Rep. at 22 (citing Mele Rep. at 2).) And "it is well settled that one expert may rely upon another expert's opinion in

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Fibrosis, 212 AJR 547, 552 (2019) (Rose Decl. Ex. 4) (noting "excellent diagnostic performance" of "key CT-based and laboratory-based biomarkers" in diagnosing cirrhosis).

formulating his own." *See In re Suboxone (Buprenorphine Hydrochloride & Naloxone) Antitrust Litig.*, MDL No. 2445, 2020 WL 6887885, at *5 (E.D. Pa. Nov. 24, 2020) ("Experts may use a mix of objective data and subjective analysis from another expert to . . . create an admissible report.") (citation omitted).

Moreover, courts have recognized that medical doctors can offer opinions relating to radiological scans, particularly where those physicians (like Dr. Mahmud) regularly review radiological imaging as part of their clinical practice. *See, e.g., In re Bauxite Containing Silica Halliday Litig. Series*, No. SX-2015-CV-097, 2024 WL 1638737, at *1-3 (V.I. Super. Ct. Mar. 26, 2024) (rejecting argument that expert was "not a radiologist" and therefore could not diagnose patient with "mixed dust pneumoconiosis" attributable to bauxite dust exposure); *In re C.R. Bard, Inc.*, 948 F. Supp. 2d 589, 624 (S.D. W. Va. 2013) (non-radiologist permitted to testify where he still "ha[d] significant experience interpreting MRIs"); *Johnson v. Sullins*, No. 5:02 CV 90, 2003 WL 26096167, at *3 (E.D. Tex. Oct. 16, 2003) (non-radiologist permitted to testify in case where he "always looks at his patients' films" as part of his clinical practice).

Dr. Mahmud testified that although he "defers" to radiologists, he regularly reviews radiological images as part of his clinical practice. (Mahmud Dep. 74:11-20, 402:14-23; *see also id.* 139:10-12 (Dr. Mahmud will do "a secondary check" of radiology reports "for my own patients just to . . . minimize the probability of

errors").) That clinical experience more than qualifies Dr. Mahmud to offer opinions based in part on references to Mr. Roberts' radiological records.[2]

B. Dr. Mahmud's Opinions Regarding Mr. Roberts' "FIB-4" Scores Are Admissible.

Dr. Mahmud's rebuttal opinion that "FIB-4" scores suggest Mr. Roberts developed cirrhosis "likely as early as November 2015" or earlier is also admissible. (Mahmud Rep. at 22.) Dr. Mahmud explains that "current best practice recommendations from major society guidance are to calculate a blood-based fibrosis score called the Fib-4, which is computed using a patient's age, AST, ALT, and platelet count." (*Id.* at 18.) By calculating this score using blood-test results, hepatologists can assess cirrhosis risks without need for an invasive liver biopsy. (*Id.*) A FIB-4 score of less than 1.3 "effectively rules out advanced fibrosis," with a greater than "90% negative predictive value." (*Id.*) On the other hand, "a Fib-4 >2.67 denotes a very high risk of advanced fibrosis." (*Id.*) According to Dr. Mahmud, Mr.

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[2] Plaintiff's argument is self-defeating because her own expert, Dr. Siddiqui, who is far less qualified than Dr. Mahmud, discusses radiological imaging throughout her own report. And unlike Dr. Mahmud (who relies on and offers opinions consistent with those of the expert radiologists in this case), Dr. Siddiqui actually ***contradicts*** Plaintiff's own radiologist's opinions that "no one could rule out the possibility" that Mr. Roberts' 2016 lesions on his liver progressed into HCC. (Dep. of Christopher Mele ("Mele Dep.") 158:22-162:3, 236:17-24, May 8, 2025 (Rose Decl. Ex. 5).) Dr. Siddiqui also characterizes Mr. Roberts' cirrhosis as "mild" in part based on her interpretation of Mr. Roberts' April 2016 CT scan (Siddiqui Rep. at 30), even though that interpretation is directly at odds with Dr. Mele's testimony that "[t]here's no way to age or grade cirrhosis on a CT" (Mele Dep. 179:22-24).

Roberts' FIB-4 score was 1.99 (above the level that would comfortably rule out cirrhosis) as early as 2009, and it soared to 3.22 by 2015, meaning "advanced fibrosis/cirrhosis likely." (*Id.* at 6-7.) Plaintiff spends 12 pages attacking Dr. Mahmud's opinion regarding FIB-4 scores, but her arguments all fail.

First, Plaintiff points to Dr. Mahmud's testimony that FIB-4 scores are not "diagnostic" tools. (Pl.'s Br. at 22 (citing Mahmud Dep. 192:25-193:12).) But as a defense expert, Dr. Mahmud is not required to affirmatively diagnose cirrhosis; rather, he need only identify "good grounds" for critiquing the "testimony by plaintiff's experts" on causation, "an issue on which plaintiff b[ears] the burden of proof." *Holbrook*, 80 F.3d at 784, 786. As Dr. Mahmud explained at his deposition, FIB-4 scores are a useful "tool to . . . risk stratify patients with chronic liver disease who may require further testing to . . . rule in or rule out cirrhosis." (Mahmud Dep. 192:25-193:12.) By explaining the history of Mr. Roberts' elevated FIB-4 scores (which long predated his HCC diagnosis in 2018), Dr. Mahmud is providing yet another reason to question Dr. Siddiqui's rejection of cirrhosis as a cause of Mr. Roberts' cancer. (Mahmud Rep. at 35.)

Second, Plaintiff is also wrong that Dr. Mahmud did not "know[] the methodology behind the FIB-4 calculation[.]" (Pl.'s Br. at 22-24.) For example, Dr. Mahmud explained that the FIB-4 formula uses "AST" and "ALT" as inputs because these items "are enzymes that are present inside liver cells," with the result that as

"[i]nflammation causes injury to liver cells . . . those AST and the ALT . . . spill out of the cells into the blood," indicating ongoing "injury to the liver[.]" (Mahmud Dep. 229:8-20.) Similarly, Dr. Mahmud explained that the formula considers platelet levels because low platelets "ha[ve] significance with respect to cirrhosis and elevated portal pressures." (*Id.* 236:6-16, 244:23-245:7.) Although Plaintiff objects to Dr. Mahmud's inputting numbers into an online calculator, MDCalc.com ("MDCalc"), in deriving Mr. Roberts' FIB-4 scores, using a calculator "does not—without significantly more" undermine an expert's opinions. *In re Fluidmaster, Inc., Water Connector Components Prods. Liab. Litig.*, MDL No. 2575, 2017 WL 1196990, at *13 (N.D. Ill. Mar. 31, 2017) (plaintiffs fail to "explain precisely how calculating PSI on a calculator, even assuming it was a rough calculation, fatally undermines the reliability of [defense expert's] conclusions"). Dr. Mahmud explained that MDCalc is "very commonly used by lots of folks, not just in hepatology, but across the field of medicine," and is "a repository for lots of prediction scores." (Mahmud Dep. 213:7-12.) And Dr. Mahmud uses MDCalc "when I see patients" (*id.* 214:7-16; *see also id.* 223:16-24 (MDCalc is a "very, very commonly used application . . . for clinicians")), confirming that he "employs in the courtroom the same level of intellectual rigor that characterizes the practice of an expert in the relevant field." *Kumho Tire Co. v. Carmichael*, 526 U.S. 137, 152 (1999).

Third, Plaintiff's argument that age is unreliably driving Mr. Roberts' elevated FIB-4 scores (Pl.'s Br. at 26), is misleading. Plaintiff quotes from Dr. Mahmud's deposition: "Q. So strictly his age is what is giving that result of fibrosis on the FIB-4 score? A. Well, yes." (*Id.* (quoting Mahmud Dep. 231:20-232:1).) But Plaintiff omits Dr. Mahmud's ***very next sentence*****:** "Well yes. I mean, ***it's a composite of all the factors you put in***." (Mahmud Dep. 232:1-3 (emphasis added).) Dr. Mahmud also explains why age is relevant: "I wouldn't expect an 18-year-old to have had sufficient time to develop advanced fibrosis and cirrhosis in the presence of MASH. It takes time to get cirrhosis." (*Id.* 232:4-21.)

Fourth, Plaintiff spends nearly seven pages criticizing Dr. Mahmud's references to platelet counts (one of several inputs in a FIB-4 score), faulting him for not considering other possible "explanations for Mr. Roberts' varying platelet counts." (Pl.'s Br. at 27.) Again, this argument seeks to reverse the relevant burden of proof, effectively demanding that Dr. Mahmud conduct a differential diagnosis to explain low platelet counts before calculating Mr. Roberts' FIB-4 score—which is ***not*** the law. *See Holbrook*, 80 F.3d at 786 (defense causation expert not held to standard of medical "certainty").

In any event, Plaintiff's complaints have no scientific basis. For example, Plaintiff criticizes Dr. Mahmud for stating that Mr. Roberts had thrombocytopenia (low platelets) in November 2015 because, while he considers low platelets to mean

a count below 150, Mr. Roberts' lab's "reference range" defined low platelets as being under 130 (his actual count was 137, between these figures). (Pl.'s Br. at 28.) But Dr. Mahmud explained that "there are well-established thresholds . . . that may differ from laboratory-reported reference ranges," and hepatologists use 150 as the cutoff for low platelets "in our guidelines." (Mahmud Dep. 99:4-17.) The National Institutes of Health agrees,[3] and the fact that Plaintiff holds a contrary view is, at most, a basis for cross-examination. Similarly, Plaintiff's contention that Dr. Mahmud did not consider "other causes" for platelet variation also rests on case reports, which are "not reliable in establishing causation." *Glastetter v. Novartis Pharms. Corp.*, 107 F. Supp. 2d 1015, 1037 n.21 (E.D. Mo. 2000), *aff'd*, 252 F.3d 986 (8th Cir. 2001). For example, although Plaintiff argues that Dr. Mahmud "was unaware that literature existed . . . demonstrating that" Proton Pump Inhibitors (which Mr. Roberts was on) "can cause thromobocytopenia" (Pl.'s Br. at 30), Dr. Mahmud explained that this "literature" consists only of "case reports" or "case series," which cannot demonstrate a "true causal association" (Mahmud Dep. 257:24-258:3). Dr. Mahmud provided similar responses when confronted with case reports addressing thiazide diuretics, such as HCTZ, and platelet counts. (*See*

[3] *See* NIH, Thrombocytopenia, https://www.nhlbi.nih.gov/health/thrombocytopenia (accessed June 5, 2025) ("A platelet count of less than 150,000 platelets per microliter is lower than normal.").

Mahmud Dep. 283:22-284:6, 285:14-287:23 (scientists cannot "extrapolate from" such "limited" data).)[4] Again, this is mainstream, accepted science; it does not undermine the admissibility of Dr. Mahmud's opinions.

For all of these reasons, Dr. Mahmud's rebuttal testimony regarding the timing of Mr. Roberts' cirrhosis is admissible.

II. DR. MAHMUD PROPERLY REBUTS DR. SIDDIQUI'S UNSCIENTIFIC CLAIM THAT NDMA CAUSED MR. ROBERTS' CIRRHOSIS.

Dr. Mahmud explains in his report why "Dr. Siddiqui's backup position that Mr. Roberts' use of NDMA-containing valsartan caused or contributed to Mr. Roberts' cirrhosis . . . is . . . unsupported by the scientific evidence." (Mahmud Rep. at 35.) As Dr. Mahmud explains, not only does Dr. Siddiqui fail to include any "citations" in support of her theory that NDMA causes cirrhosis in humans, but "a search of the scientific literature and reading of major hepatology society guidelines did not identify any high-quality data in human subjects demonstrating" such a relationship. (*Id.*) This rebuttal testimony is admissible because it has "good grounds" and "could help the jury to evaluate testimony by plaintiff's expert[.]" *Holbrook*, 80 F.3d at 784-86.

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[4] Plaintiff speculates that NDMA in valsartan must be the cause of Mr. Roberts' low platelets on the ground that his count supposedly "rebounded" soon after he stopped taking valsartan potentially contaminated with NDMA. (Pl.'s Br. at 32.) But his count increased by a mere "15 or 16 points"—to 126, which Dr. Mahmud explained is still low by any measure. (Mahmud Dep. 337:1-16.)

Plaintiff nevertheless seeks to exclude this opinion on the ground that Dr. Mahmud failed to consider "reliable scientific evidence that NDMA ***can*** cause cirrhosis in humans[.]" (Pl.'s Br. at 35 (emphasis added).) But what Plaintiff labels "reliable scientific evidence" is actually a series of "statements from the United States Health and Human Services detailing how two researchers got liver cirrhosis after exposure to NDMA in a laboratory setting." (*Id.*) When confronted with this anecdotal case report, Dr. Mahmud appropriately explained why it cannot support a "causal link" between NDMA and cirrhosis in humans. (Mahmud Dep. 366:17-21.) For example, the two individuals "could have already had cirrhosis from a multitude of different causes" and/or might have had any number of other exposures not reflected (much less controlled for) in the case report. (*Id.* 366:8-16.) In short, this testimony highlights why ***Plaintiff's*** expert's causation opinion is inadmissible, *see Glastetter*, 107 F. Supp. 2d at 1037 n.21 (case reports are "not reliable"); it does not support excluding Dr. Mahmud's rebuttal.

III. DR. MAHMUD'S OPINION CRITICIZING DR. SIDDIQUI FOR FAILING TO PROPERLY CONSIDER MR. ROBERTS' MASH AS A CAUSE OF HIS HCC IS RELIABLE.

Dr. Mahmud explains in his report that MASLD and MASH (previously known as NAFLD and NASH, respectively) "increase the risk of HCC in two ways": (1) through the development of cirrhosis; and (2) as an independent risk factor. (Mahmud Rep. at 23.) As Dr. Mahmud explains, "Dr. Siddiqui . . . lacks a scientific

basis to rule out MASLD/MASH as an independent cause of Mr. Roberts' HCC" because she unscientifically lumps all of Mr. Roberts' non-valsartan-related cancer risk factors together as contributing to cirrhosis and "ignores the fact that many patients with MASLD/MASH may develop HCC in the absence of cirrhosis[.]" (*Id.* at 38.) This rebuttal testimony, supported by numerous citations to scientific literature and Mr. Roberts' own medical records (*see, e.g., id.* at 23-24), is sufficiently well-grounded to be admissible under Rule 702. *See Holbrook*, 80 F.3d at 784-86.

Plaintiff urges the Court to exclude this rebuttal testimony because "Mr. Roberts was never diagnosed with NASH, showed no symptoms of NASH, and never underwent a biopsy necessary to diagnose NASH[.]"[5] (Pl.'s Br. at 21.) But "MASLD/MASH" is "consistently reflected in the problem list of [Mr. Roberts'] treating providers throughout his medical history[.]" (Mahmud Rep. at 24.) As early as 2009, Mr. Roberts' treating physician, Dr. Ives, noted that Mr. Roberts "ha[d] been told over the years that he probably has a fatty liver, ***i.e., NASH***." (GRobertsJr-CA-000661(emphasis added) (Rose Decl. Ex. 6).) Mr. Roberts' treating physicians also characterized his HCC as "arising out of NASH cirrhosis[.]" (GRobertsJr-UABHIM-MD-000085 (Rose Decl. Ex. 1).)

[5] As previously noted, NASH (non-alcoholic steatohepatitis) was the prior nomenclature used for MASH.

Contrary to Plaintiff's claim, Dr. Mahmud did not testify that a biopsy is "necessary" to "confirm a diagnosis of NASH[.]" (Pl.'s Br. at 21.) What he said is that while a biopsy might be required "from a . . . very technical standpoint" "if you're being a medical purist . . . these days we do not commonly perform liver biopsy." (Mahmud Dep. 159:14-160:20.) Instead, "[w]e are able to generally infer if a patient has NASH or MASH without subjecting them to a liver biopsy" using "evidence from their blood work, for instance, and their fibrosis or their FIB-4 score[.]" (*Id.*) Again, Dr. Mahmud is not alone in this thinking; rather, his position aligns with that of the broader hepatologic community.[6] For this, reason, too, Plaintiff's arguments should be rejected.

IV. DR. MAHMUD HAS GOOD GROUNDS FOR HIS OPINIONS REGARDING ANIMAL STUDIES.

Plaintiff does not challenge any of the detailed statements in Dr. Mahmud's report as to why animal studies addressing NDMA and liver cancer "cannot be assumed to extrapolate directly to humans," including his explanation of the "key differences in metabolic pathways, exposure levels, and disease biology between animal models and humans." (Mahmud Rep. at 26.) Plaintiff instead takes issue with

[6] *See* Fasiha Kanwal et al., *Metabolic dysfunction–associated steatotic liver disease: Update and impact of new nomenclature on the American Association for the Study of Liver Diseases practice guidance on nonalcoholic fatty liver disease*, 79 Hepatology 1212, 1216 (2024) (Rose Decl. Ex. 7) (noting that although "MASH . . . traditionally required a liver biopsy . . . clinical practice has shifted away from biopsy toward using biomarkers to noninvasively categorize MASLD severity").

an "additional" observation Dr. Mahmud offered at his deposition in responding to counsel's questioning—i.e., that it is extremely difficult to extrapolate from animal data because the animals "have normal livers at baseline." (Mahmud Dep. 372:1-8.) As he explained, "if you have a liver that already has cirrhosis . . . [i]t is not as effective as a healthy liver in metabolizing NDMA to the toxic metabolites," which allegedly cause the cancer. (*Id.* 372:21-25; *see also id.* 372:9-20 (the postulated mechanistic theory necessarily assumes that NDMA is "activated in the liver").)

Plaintiff tries to twist this testimony into a completely new opinion that "someone with pre-existing liver cirrhosis/disease would be at a decreased risk of liver cancer from NDMA exposure." (Pl.'s Br. at 36.) But that is Plaintiff's extrapolation, not Dr. Mahmud's. Instead, all he said was that "you have to be very cautious in translating animal study findings to humans," and that such caution includes consideration of the fact that the animal studies relied upon by Plaintiff's experts involved a "healthy liver" (i.e., without cirrhosis). (Mahmud Dep. 371:22-372:7.) As Dr. Mahmud explained, "we [hepatologists] actually change the way we prescribe certain medications if we think cirrhosis is present," precisely because a medicine requires metabolic reactions to work. (*Id.* 372:21-373:5.) The "same principle applies" to NDMA, which needs to be metabolized to have Plaintiff's experts' postulated relevant biological effect on the liver. (*Id.* 389:24-390:6.) This reasoning, based on Dr. Mahmud's extensive knowledge of liver function, easily

satisfies Rule 702.

V. DR. MAHMUD HAS GOOD GROUNDS FOR OPINING THAT MR. ROBERTS MAY WELL HAVE HAD HCC PRIOR TO HIS VALSARTAN USE.

Dr. Mahmud also criticizes Dr. Siddiqui for reaching a causal conclusion despite the short period (23 months) between Mr. Roberts' first exposure to valsartan potentially contaminated with NDMA and his HICC diagnosis. (Mahmud Rep. at 33.) As he explains, this opinion is particularly problematic because Mr. Roberts may well have had HCC before ever taking a single valsartan pill potentially contaminated with NDMA. Plaintiff's criticisms of this opinion only highlight the problems in her own expert's opinions.

A. Dr. Mahmud's "Doubling Time" Opinions Are Admissible.

As Dr. Mahmud explains, the literature on tumor "doubling time" suggests that Mr. Roberts' tumor was almost certainly already growing before September 2016, when he first took valsartan potentially contaminated with NDMA. (Mahmud Rep. at 33.) In so opining, Dr. Mahmud relies on a meta-analysis, finding that the doubling time for such tumors ranges from 3.9 to 5.3 months (using a 95% confidence interval), with an average of 4.6 months. (*Id.*) Dr. Mahmud uses these data to "estimate historic tumor sizes" by calculating tumor volume and diameter, all of which indicate that Mr. Roberts likely "had undiagnosed HCC at the time of his first exposure to" valsartan potentially contaminated with NDMA. (*Id.* at 33-34.)

Plaintiff's challenges to this calculation are meritless.

First, Plaintiff argues that to perform his calculations, "Dr. Mahmud had to assume that Mr. Roberts' liver cancer was spherical when it started and remained spherical throughout its progression." (Pl.'s Br. at 12.) However, as Dr. Mahmud explained at his deposition, that is a "fairly standard assumption" because even if cancers are not perfect spheres, they "tend to grow roughly in a[] spherical shape." (Mahmud Dep. 394:5-18.) The scientific literature further supports this assumption.[7] Plaintiff does not cite any scientific authority embracing her contrary position, and if she has any, her counsel can pursue it through cross-examination of Dr. Mahmud. *See Dzielak v. Whirlpool Corp.*, No. 2:12-0089 (KM) (JBC), 2017 U.S. Dist. LEXIS 39232, at *28 (D.N.J. Mar. 17, 2017) ("methodological assumption" "may be challenged on cross-examination").

Second, Plaintiff also criticizes Dr. Mahmud for using a doubling-time rate of 3.9 months for the most aggressive scenario in his table, even though one of the studies addressed in the meta-analysis cited by Dr. Mahmud "notes that liver tumors can double every 2.2 months." (Pl.'s Br. at 15.) However, the doubling times used

[7] *See, e.g.*, Jong Kwan Kim et al., *Tumor volume doubling time as a dynamic prognostic marker for patients with hepatocellular carcinoma*, 62 Digestive Diseases & Sciences 2923 (2017) (assuming spherical shape) (Rose Decl. Ex. 8); Chansik An et al., *Growth rate of early-stage hepatocellular carcinoma in patients with chronic liver disease*, 21 Clinical & Molecular Hepatology 279 (2015) (similar) (Rose Decl. Ex. 9).

by Dr. Mahmud reflect the average (4.6 months), as well as the outer bounds (3.9 and 5.3 months) of a 95% confidence interval of HCC tumor growth. (Mahmud Rep. at 33 (citing Nathani et al., *Hepatocellular carcinoma tumour volume doubling time: a systematic review and meta-analysis*, 70 Gut 401 (2021)).) Dr. Mahmud thus adhered to basic statistical principles of "mov[ing] from a single value estimate . . . to a range of values that are considered to be plausible for the population."[8]

Third, Plaintiff argues that Dr. Mahmud did not "understand the tumor volume doubling time calculation performed or to what extent it can be applied." (Pl.'s Br. at 16.) Neither is true. With respect to the former claim, Plaintiff merely highlights testimony referencing "'the formulas that [Dr. Mahmud] laid out'" in his report. (*Id.* (citing Mahmud Dep. 407:12-15).) And with respect to the latter claim, Plaintiff points to testimony by Dr. Mahmud that he was "'not immediately sure'" if it would be possible to back-calculate to the very inception of a patient's cancer or capture tumors smaller than 1 centimeter. (*Id.* at 17 (citing Mahmud Dep. 409:6-14).) This critique is irrelevant because Dr. Mahmud is not purporting to divine when Mr. Roberts first developed HCC. And in any event, the remainder of Dr. Mahmud's testimony makes clear that the doubling-time methodology would not be appropriate

[8] Martin J. Gardner & Douglas G. Altman, *Confidence intervals rather than P values: estimation rather than hypothesis testing*, 292 Br. Med. J. 746 (1986) (Rose Decl. Ex. 10).

for tumors "less than .1 centimeters" because "you have to be able to measure it on imaging." (Mahmud Dep. 409:15-22.)

Fourth, Plaintiff argues that, according to Dr. Mahmud's testimony, Mr. Roberts should have had a "2-centimeter" tumor in April 2016, rather than the smaller lesions that were present on the 2016 CT scan. (Pl.'s Br. at 13.) However, a typical range of tumor-growth doubling times back-dated from Mr. Roberts' August 2018 diagnosis would actually predict only 1.15-cm tumors in April 2016, not much larger than those actually visible on the CT scan. (Mahmud Dep. 400:7-401:6.) Regardless, Dr. Mahmud fully acknowledged that there is "variation" in doubling times and that Mr. Roberts' lesions in April 2016 may have been slightly larger or smaller than the estimate. (*Id.*)

Fifth, and finally, Plaintiff relies on testimony from Defendants' expert cancer biologist, Dr. Lewis Chodosh, stating that while doubling-time analyses are "generally" used in the "research setting or human studies setting," they are "not typically something that's done on an individual patient basis." (Pl.'s Br. at 18 (citing Chodosh Dep. 86:19-92:20 (Pl.'s Br. Ex. D)).) But Plaintiff omits Dr. Chodosh's explanation that he simply could not "think of a clinically relevant reason for a patient" and doctor to discuss this issue. (Chodosh Dep. 88:6-12 (counsel: "Why wouldn't it serve a purpose?").) Here, by contrast, there is an obvious purpose for the doubling-time calculations—i.e., to specifically rebut Dr. Siddiqui's claim that

NDMA in valsartan caused Mr. Roberts' HCC to grow so fast that it somehow reached "Stage III" not long after Mr. Roberts used valsartan potentially contaminated with NDMA. (Siddiqui Rep. at 29-31.) Dr. Chodosh recognized later in his deposition that scientists "can use . . . data" from studies on tumor volume doubling time to "infer what is the minimum latency for" a particular cancer. (Chodosh Dep. 235:22-236:6; *see also id.* 236:7-17 (testifying that it is entirely proper to "tak[e] a population estimate of tumor volume doubling time and then apply[] it to a patient sort of in . . . [a] retroactive manner").)

Again, Dr. Mahmud's "rebuttal evidence is properly admissible" because "it will explain, repel, [and] counteract" the opinions being offered by Plaintiff's expert. *FTC v. Innovative Designs, Inc.*, No. 2:16-cv-01669-NBF, 2018 WL 3611510, at *3 (W.D. Pa. July 27, 2018).

B. Dr. Mahmud Also Appropriately Relied On Dr. Chernyak's Opinion That Mr. Roberts' 2016 CT Scan Showed "LR-3" Lesions.

Dr. Mahmud's doubling-time projections are also "consistent" with the radiological findings of defense expert Dr. Chernyak regarding Mr. Roberts' April 2016 CT scan. (Mahmud Rep. at 34.) In particular, Dr. Chernyak noted three LR-3 lesions (i.e., intermediate probability of malignancy), measuring 0.6 cm (segment VII), 0.5 cm (segment V/VIII), and 0.5 cm (segment VI). (*Id.* (citing Chernyak Rep. at 5 & Exhibit B).) As Dr. Mahmud explains, Dr. Chernyak's observation that the segment V/VIII lesion identified in 2016 is in the same location as one of the LR-5

lesions (i.e., definite HCC) identified in the 2018 MRI is "[o]f critical importance" because that means it "cannot be ruled out that this LR-3 lesion represented HCC, which subsequently grew to become the LR-5 lesion[.]" (*Id.*)

Plaintiff argues that Dr. Mahmud "lack[s] the radiological expertise" to discuss Dr. Chernyak's radiological findings (Pl.'s Br. at 11), but as previously discussed, "it is well settled that one expert may rely upon another expert's opinion in formulating his own." *In re Suboxone*, 2020 WL 6887885, at *5 (citation omitted). Such reliance is all the more appropriate here because Dr. Mahmud both regularly "review[s] [images] with the radiologist" and is "familiar with the LI-RADS criteria" applied by Dr. Chernyak. (Mahmud Dep. 74:11-20, 402:14-23; *see also id.* 139:10-12 (Dr. Mahmud will "do this as a secondary check" of radiology reports "for my own patients just to . . . minimize the probability of errors.").) *See Bell v. Mine Safety Appliances*, No. 1:13-cv-01075, 2016 WL 705214, at *2 (W.D. Ark. Jan. 26, 2016) (non-radiologist appropriately relied on radiology report "to form his opinion").[9]

Plaintiff's complaints regarding Dr. Mahmud's reliance on Dr. Chernyak's

[9] Dr. Mahmud's opinions are also consistent with the opinions of Mr. Roberts' treating radiologist in 2016, who recommended additional screening after his April 2016 scans, precisely because Mr. Roberts' pre-existing lesions could have had "[b]enign or malignant etiologies[.]" (GRobertsJr-TH-MD-000944 (Rose Decl. Ex. 11).)

LR-3 opinion are just thinly veiled attacks on Dr. Chernyak, a prominent radiologist at Columbia University. As explained in greater detail in Defendants' opposition to Plaintiff's motion to exclude Dr. Chernyak, Dr. Chernyak was able to observe all of the major features of LI-RADS on Mr. Roberts' 2016 CT, which was sufficient for her to characterize the observed lesions as LR-3. (Chernyak Rep. at 5; *see also* Chernyak Dep. 54:11-24; Opp'n to Mot. to Exclude Chernyak at 9-20.) Accordingly, there are "good grounds" for Dr. Chernyak's application of the LI-RADS criteria and, by extension, Dr. Mahmud's reliance on it in his report.[10] *See Holbrook*, 80 F.3d at 784-86. For this reason, too, Plaintiff's attacks on Dr. Mahmud's opinions should be rejected.

VI. DR. MAHMUD HAS GOOD GROUNDS FOR OPINING AS TO THE CAUSE OF MR. ROBERTS' LIVER CANCER.

At the very end of her brief, Plaintiff includes a final argument under the heading "Dr. Mahmud Can Not Reliably Opine as to the Cause of Mr. Roberts' Liver Cancer[.]" (Pl.'s Br. at 37.) According to Plaintiff, "Dr. Mahmud did not properly consider Mr. Roberts' NDMA exposure as a potential cause for his liver cancer, because Dr. Mahmud had already . . . concluded that Mr. Roberts had liver cancer

[10] Although Plaintiff highlights Dr. Chernyak's stray reference to a "nonexistent" 2016 MRI (Pl.'s Br. at 10), Dr. Chernyak explained that was a typographical error, and that she intended to refer to a CT scan (Chernyak Dep. 25:18-19), as she did approximately two dozen times in her report (*see generally* Chernyak Rep.).

prior to ingesting" valsartan potentially contaminated with NDMA. (*Id.* at 38.) The Court should reject this argument for several reasons. As an initial matter, Plaintiff is once again seeking to engraft onto Dr. Mahmud "a burden which the defense d[oes] not bear"—i.e., to affirmatively ***disprove*** Plaintiff's NDMA-based theory of causation. *See Holbook*, 80 F.3d at 786. Instead, it is entirely appropriate for a defense rebuttal expert to point out that other potential causes "could not be excluded" because doing so "help[s] the jury to evaluate testimony by plaintiff's experts" with respect to "an issue on which plaintiff bore the burden of proof." *Id.*

In any event, Dr. Mahmud thoroughly considered and ultimately rejected Mr. Roberts' NDMA exposure as a potential cause of his HCC. Over the course of seven pages in his report, Dr. Mahmud thoroughly explains why "[t]here is no established scientific basis upon which to conclude that Mr. Roberts' valsartan use was a possible cause of his HCC." (Mahmud Rep. at 25-32.) First, Dr. Mahmud goes through the major national and international hepatology society guidelines for HCC. As Dr. Mahmud recounts, none of those organizations lists NDMA or valsartan as a risk factor for HCC, whereas all of them do recognize a number of risk factors that apply to Mr. Roberts (e.g., cirrhosis, MASLD/MASH, obesity, and diabetes). (*Id.* at 25-26.) Second, Dr. Mahmud explains why "Dr. Siddiqui's suggestion that NDMA exposure has been clearly linked to liver cancer in humans . . . is incorrect," noting that she rests primarily on inapposite animal studies and an occupational health study

by Hidajat that has "major limitations[.]" (*Id.* at 26, 30.) And third, Dr. Mahmud addresses the limited epidemiological studies addressing NDMA in valsartan and liver cancer in painstaking detail, explaining that they detected, at most, a "weak" association that is "dwarfed by the other strong and well-established risk factors for HCC" just discussed and do not support an inference of causation under the Bradford Hill framework. (*Id.* at 3, 27-32.)

Plaintiff does not address any of this detailed analysis. Instead, she focuses solely on Dr. Mahmud's testimony regarding the timing of Mr. Roberts' HCC—i.e., whether the cancer arose before Mr. Roberts first took valsartan potentially contaminated with NDMA. (Pl.'s Br. at 37.) As already discussed, Dr. Mahmud has sufficient grounds for criticizing Plaintiff's own expert's failure to properly consider the possibility that Mr. Roberts had HCC before he started taking valsartan potentially contaminated with NDMA in 2016. But even if those opinions were inadmissible, that would have zero bearing on the remainder of Dr. Mahmud's causation analysis, which in no way turns on the precise timing of Mr. Roberts' cancer development. Rather, as Dr. Mahmud's report makes clear, his opinions are based on his own independent review of the literature, Mr. Roberts' medical history, and Dr. Siddiqui's disregard of that existing evidence and elementary scientific principles. (*See* Mahmud Rep. at 25-42.)

In sum, Plaintiff has not come close to justifying exclusion of any portion of Dr. Mahmud's causation opinion, much less the entirety of it.

CONCLUSION

For the foregoing reasons, the Court should deny Plaintiff's motion to exclude the opinions of Dr. Mahmud.

Dated: June 26, 2025

Respectfully submitted,

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CERTIFICATE OF SERVICE

I HEREBY CERTIFY that on June 26, 2025, a true and correct copy of the foregoing document was served upon counsel of record via operation of the Court's electronic filing system.

Dated: June 26, 2025

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